Dermatology of breeds (Retrievers, Shar-pei, Chow-chow)

Rostaher, A

DERMATOLOGY OF BREEDS

Dermatology day of The Slovene Dermatology Study Group
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Generalni sponzor:

zoetis

Dear friends and colleagues,

For the Slovene Veterinary Dermatology Study Group it is a great pleasure to welcome you to our yearly dermatology day. The topic of the day is the ***Dermatology of breeds***. The main features of the most common skin diseases in certain breeds of dogs will be presented. Diseases unique to a breed will be discussed in more detail and we tried to pick up the most interesting ones, hopefully important for everyone's daily clinical practice. Finally, we planned to devote some time to case discussions. Therefore, please feel free to share your cases with every body. We would love our lectures to be as much as possible interactive.

Mihaela Bizjak, Rostaher Ana and SZVMŽ board

Speaker information

Svetlana Belova, DVM, DipECVD

Svetlana Belova graduated from the Faculty of Veterinary Medicine, Estonian Agricultural University in 1995 and worked in small animal practice before starting ECVD (European College of Veterinary Dermatology) residency programme at the Veterinary School in Zurich, Switzerland in 2007. After 4 years of training and succesful examination in 2011 she became Diplomate of the ECVD. Svetlana is a lecturer in dermatology and involved in clinical teaching of veterinary students at the Estonian University of Life Sciences since 2005.

She gave more than 70 presentations and workshops in dermatology for veterinary practitioners in Estonia, Russia, Belorussia, Ukraine, Latvia, Lithuania, Finland, Hungary and Poland. Apart from teaching she is providing referral dermatology service in small animal practices in Tallinn and Helsinki. In 2010 was awarded by the RSAVA (Russian Small Animal Veterinary Association) with "The Golden scalpel – lecturer of the year" and by the EVA (Estonian Veterinary Association) with the title "Veterinarian of the year".

Ana Rostaher, dr.vet.med., dipl ECVD

Ana graduated from the Slovene veterinary school in Ljubljana in 2002. She spent 4 years working in a small animal practice in Slovenia and finished an internship at the Veterinary University in Vienna in 2005. During her veterinary dermatology residency at the Department of Small Animal Medicine, Veterinary School Munich, Ana finished also a research externship at the Jackson Laboratory Maine, Bar Harbour in 2010. She became Diplomate of the European College of Veterinary Dermatology in 2011 and worked as veterinary dermatology consultant in two private veterinary clinics in Paris area. Currently she is employed at the Veterinary School in Zurich, Switzerland as senior clinician and prepares her habilitation in the field of veterinary allergology. She gave more than 50 presentations for veterinary practitioners and students and published over 60 publications. Ana is currently secretary of the European Society of Veterinary Dermatology, Credential Committee Member of the European College of Veterinary Dermatology and President of the Dermatology Study Group in Slovenia.

Timetable

8.30 -	Registration
09.00 - 10.30	German Shepherd, Bernese Mountain Dog, Nordic breeds (S Belova)
10.30 - 11.00	Coffee break
11.00 - 12.30	Retrievers, Shar-pei, Chow-chow (A Rostaher)
12.30 – 13.00	Lecture sponsor
13.00 - 14.30	Lunch break
14.30 – 16.00	English bulldog, French bulldog, Bull Terrier, Cavalier King Charles, Pointer (S Belova)
16.00 – 16.15	Coffee break
16.15 – 17.00	Case discussions
17.15 – 17.45	Annual General Meeting of the Slovene Dermatology Study Group

Svetlana Belova, DVM, DipECVD

German Shepherd Dog (GSD)

Symmetrical lupoid onychodystrophy (SLO) is an immune-mediated claw disease characterized by onychomadesis and regrowth of deformed claws. Young adult to middle-aged GSD appear predisposed. The exact etiology is not known.
There is usually an acute onset of nail loss and associated pain/lameness. Initially, one or two claws are lost, but over a course of a few weeks all claws become affected. Regrown claws are misshapen, soft or brittle, and often slough again. Affected dogs are otherwise healthy. Secondary bacterial paronychia is common.
Usually a diagnosis of SLO can be made based on history and clinical signs, however a biopsy of the digit can be required for definitive diagnosis.
Initial aggressive treatment with immunosuppressive doses of glucocorticosteroids (GCS) is often needed. Secondary bacterial paronychia is treated with appropriate systemic antibiotics. For maintenance therapy oral fatty acids, oral vitamin E, doxycycline/niacinamide, and pentoxifylline are commonly used. Food allergy has been identified in a few cases of SLO, so it may be appropriate to go through a hypoallergenic food trial. A number of dogs have also been diagnosed with hypothyroidism at the same time SLO has been diagnosed. A full thyroid panel may be recommended.
Prognosis for nail regrowth is good, although some nails may remain deformed. Usually long term maintenance therapy is needed to maintain remission.

Perianal fistulas (PAF) is a chronic, painful, progressive inflammatory
and ulcerative disease associated with the perianal, anal, and/or perirectal tissues with fistulous tracts as a characteristic feature. PAF usually affects middle-aged GSD. No sex predilection is reported.
Definitive cause of PAF is not known, but a multifactorial immune-mediated disease is suspected. Lesions may vary from superficial pinpoint tracts to large ulcerated areas. Tracts may interconnect and/or go deep into surrounding tissue and occasionally communicate with the rectum, anus, and/or anal sacs. Fibrosis of the anus and rectum is common. Other possible signs are tenesmus, dyschezia, hematochezia, constipation or diarrhoea, perianal licking. Diagnosis is based on history, clinical findings, and ruling out other diagnostic differentials (mainly neoplasia) with help of cytology and histopathology.
Medical management involves use of immunosuppressive drugs: cyclosporine +/ GCS or +/-azoles, GCS +/-azathioprine, topical GCS and tacrolimus. Careful bathing/cleaning, systemic and local antimicrobials are administered for treating secondary infections. A hypoallergenic food trial is recommended since many cases may have an underlying allergic cause. In severe cases, surgery to debride or remove as much infected tissue as possible may be beneficial. Surgery has a high risk of potential complications and is used only in cases where medical treatment fails. PAF is a frustrating to treat and prognosis is guarded.

Metatarsal fistulation of the German Shepherd Dog is a rare condition characterized by the formation of deep draining tracts in the skin of metatarsal area.
The cause and mechanism of disease are unknown, but an autoimmune disease may be responsible, as circulating antibodies against collagen type I and II were elevated in some affected dogs. Strong breed predisposition suggests heritability.
Initially there is a soft swelling just proximal to the metatarsal footpad which progress to single or multiple well demarcated sinus tracts with oozing serosanguineous discharge. Usually both hindlimbs are affected and occasionally fistulas occur in the plantar metacarpal skin. Lesions are not pruritic, but mild pain is possible. Chronically affected skin may become scarred.
Cytological examination of the discharge is characteristic for pyogranulomatous inflammation. Bacterial culture of the discharge is sterile, although secondary bacterial infection of lesions may occur.
Histopathology: severe inflammatory reaction (neutrophils, macrophages, lymphocytes, plasma cells and multi-nucleated giant cells) mostly involving the panniculus.

Differential diagnoses: foreign bodies, puncture wound, deep bacterial or fungal infection. Diagnosis is based on anamnesis, clinical findings and ruling out other differentials. Condition is often chronical and life-long treatment is needed. Treatment options:

- 0.1% tacrolimus 2xdaily
- Doxycycline (500 mg) and niacinamide (500 mg) 3xdaily
- Prednisolone 1-2mg/kg/day, then tapered to a maintenance regimen
- Vitamin E 300 IU 2xday
- Cyclosporine 5mg/kg/day

Discoid lupus erythematosus (DLE) is one of the most common autoimmune skin disorder in dogs, which particularly affects "long-nosed" breeds and GSD among them. DLE is differentiated from systemic lupus erythematosus (SLE) by the absence of multisystemic manifestations and negative anti-nuclear antigen (ANA) titer. In DLE IgG autoantibodies deposited at the basement membrane zone.
Most common symptom is erythema, depigmentation, erosions and loss of cobblestone texture of the nasal planum. Similar bilateral symmetrical lesions may also develop in the periocular region, on the ear pinnae, and on the lips. Rarely, lesions may be found on the trunk, limbs, footpads, genitals, and the perianal regions. Lesions can be aggravated by ultraviolet light exposure.
Definitive diagnosis is based on histopathology results with typical interface changes. Therapeutic options include avoiding sun exposure and use of sunscreens, oral vitamin E, oral niacinamide and doxycycline, topical glucocorticosteroids (GCS) and tacrolimus. More refractory cases may need systemic GCS+/- azathioprine or chlorambucil, or cyclosporine. Treatment is often life long, but there is a good prognosis for long-term remission.

Bernese Mountain Dog

This breed has strong predisposition to some histiocytic proliferative diseases, which occur as inflammatory or neoplastic processes with immune dysregulation.
Several histiocytic proliferative diseases have been recognized in dogs:

- Cutaneous histiocytoma
- Cutaneous Langerhans cell histiocytosis
- Reactive cutaneous histiocytosis (RCH)
- Reactive systemic histiocytosis (RSH)
- Histiocytic sarcoma complex (HSC)

Bernese Mountain dog is predisposed to the last two listed.

Reactive histiocytoses (RCH and RSH) are related disorders arising from activated interstitial dendritic cells (DC). Both primarily target the skin and subcutaneous tissue. Lesions of RCH are limited to the skin, while RSH affects also other organs, such as lymph nodes, eyes, nasal cavity, lungs, spleen and bone marrow.
Reactive CH predominately affects young to middle-aged Bernese Mountain dogs (2–8 years). Solitary, but most often multiple non-pruritic and non-painful, haired or partially alopecic, nodules and plaques are seen on the skin of the head (especially nasal apex, nasal planum, and eyelids), neck, perineum, scrotum, and extremities. Lesions may occur in a linear fashion, arranging along lymphatics or blood vessels. Necrosis and crateriform ulcerations are possible. Other clinical features depend on the organ systems affected. Nodular or diffuse swelling of the mucous membranes of the nares and nasal cavity is associated with respiratory stertor. If eyes are affected - bilateral conjunctivitis with marked chemosis and scleritis are common, intrabulbar and retrobulbar lesions may occasionally occur. Lymphadenopathy may be observed. Scrotal lesions may be accompanied by orchitis. Additional lesions may occur in the lungs, spleen, liver and bone marrow.

Etiology of reactive histiocytosis is not clear. Lesions are consistent with a persistent reactive inflammatory process in response to antigen(s) stimulation, which suggests an ineffective down-regulation of the inflammation. Extensive evaluations for possible responsive infectious antigens have been unsuccessful. Marked Bernese Mountain dog predilection for RSH supports at least partial genetic basis. Diagnosis is confirmed by histopathology.
Although spontaneous regressions may occur, majority of cases follow a slowly progressive waxing and waning course. With treatment the dogs have a rather good prognosis. First choice is cyclosporin in high doses (up to 10-25mg/kg/day). Ocular lesions require additional topical 2% cyclosporin eye drops. Good alternative to cyclosporine is leflunomide administered at 4 mg/kg/d. Glucocorticosteroids may not be successful.

Histiocytic sarcomas (as reactive histiocytoses) mostly arise from interstitial DC, which occur in almost all tissues. HS may arise as single or multiple lesions in one organ (spleen, skin, lung, lymph node, bone marrow, brain, articular tissue of large limb joints), but very often this so called localized form (LHS) will rapidly spread to involve multiple organs. Last form of the disease was formerly called malignant histiocytosis (MH) but is now more appropriately termed disseminated HS (DHS). The age of affected dogs ranges from 2 to 13 years. A female-male ratio of 1,2:1 has been observed in Bernese mountain dogs. Pedigree analyses support a polygenic mode of inheritance in Bernese Mountain Dogs. Recently, studies of the genomic loci involved in HS in Bernese Mountain dogs have found abnormalities in tumor suppressor gene loci (CDKN2A/B , RB1, and PTEN) that are similar to those described in human HS. Clinical signs of HS are non-specific and include anorexia, weight loss and lethargy. Other signs will depend on the organ(s) involved. For example, lung neoplasia leads to cough and dyspnea, central nervous system (CNS) involvement leads to seizures, incoordination and paralysis, articular HS leads to lameness. Also dogs often have mild, nonregenerative anemia.
Diagnosis based on histopathology.
LHS are locally aggressive neoplasms with great potential for metastases. LHS of the skin and subcutis are recognized relatively early. This enables early therapeutic attempts - more favourable outcome is observed with wide surgical excision. Follow-up with radiation therapy should also be considered. Evaluation of draining lymph nodes, abdominal ultrasound and chest radiographs should be preformed before surgical or radiation therapy is attempted.
DHS has a poor prognosis as no effective treatment is known.

Nordic breeds

Zinc-responsive dermatosis seen in northern breed dogs (Siberian husky, Alaskan malamute, Samoyed) is a scaling and crusting skin disease resulting from inherited impairment in the absorption of zinc. Zinc is important for a variety of biological functions, including regulation of the immune response, modulation of keratogenesis and wound healing, maintenance of normal reproductive function, and acuity of taste and smell. Evidence indicates that cellular (keratinocytic) response to free radical-induced oxidative stress is involved in the pathogenesis of skin lesions in zinc-responsive dermatosis.
Clinical signs usually become apparent during the first year of life. The most commonly encountered skin lesions are erythema, alopecia, scales and crusts that primarily affect the head (periocular areas, dorsal aspect of the muzzle, nasal planum, lips) but also mucocutaneous junctions and pressure points (elbows, hocks, footpads). Pruritus ranges from absent to very severe. Secondary infections often complicate the dermatitis. In severe cases, anorexia, lethargy, retarded growth, suppurative paronychia, swollen paws, and peripheral lymphadenopathy are seen.
Diagnosis is based on the signalment, clinical signs and skin histopathology results. Zinc concentrations in serum have only a corroborative value in the diagnosis.
Main treatment consists of lifelong oral zinc supplementation. Initial dose of 2-3 mg/kg is recommended. Some cases necessitate intravenous injection of zinc sulfate (10 mg/kg), at least in the beginning of therapy. Additional therapy includes glucocorticosteroids, fatty acids, bathing with keratolytic shampoos, and antimicrobial therapy if needed. Currently, there is no genetic (DNA) test available. Affected dogs should not be bred.


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Uveodermatologic syndrome (also called Vogt-Koyanagi-Harada-like syndrome) is an autoimmune condition affecting melanocytes (specifically tyrosinase and related proteins) and characterized by chronic granulomatous inflammation of eye tissues and skin. The syndrome occurs most commonly in the Nordic breeds such as the Akita (around 80% of cases!), Samoyed and Siberian Husky. Recent report describes increased the risk for developing of UDS in American Akitas with the DQA1*00201 allele, further supporting a genetic component of the disease. Age of onset is from 6 months to 6 years, with a slightly increased incidence in males. Ophthalmic signs usually precede dermatologic ones and include uveitis with secondary changes such as glaucoma, cataracts, bullous retinal detachment, and blindness. Skin lesions predominantly affect the face (bridge of the nose/ nasal planum, periocular regions and lips) and may include leukotrichia and leukoderma with erythema, ulceration and crusting of variable severity. Lesions can also develop on the scrotum or vulva, anus, footpads, pinnae and within the oral cavity.
Diagnosis is based on signalment, characteristic clinical signs and skin histopathology.
Treatment mainly consists of a combination of systemic immunosuppression (systemic glucocorticosteroids+/-azathioprine, cyclosporine) and topical ophthalmic anti-inflammatory medications.
Prognosis in general is good with regard to the skin disease but the long-term prognosis is poor with regard to vision.
There is no DNA test available.

Sebaceous adenitis (SA) is a suspected immune-mediated disease that targets and destroys sebaceous glands and characterized by hyperkeratosis and alopecia. Akita, samoyed and possibly also the chow-chow are predisposed among Nordic breed dogs. Autosomal recessive mode of inheritance is suggested for Akita. There is no sex predilection. Clinical signs most commonly appear in young adult to middle-aged dogs and include varying degree of alopecia and scaling with follicular casts as a distinctive feature. Lesions are symmetrical and commonly start dorsally on the head, neck and pinnae and proceed caudally and later involve the trunk. So called "rat tail" can be seen. Pruritus is usually not a feature, but can develop in case of secondary pyoderma. Otitis externa with dry, dark adherent scales in the ear canal is a common feature.
For definitive diagnosis skin histopathology result is required. No DNA test available.
Most effective treatment consists of combination of topical therapy (antiseborrheic shampoos, oil soaks and humectant sprays) and systemic cyclosporine 5 mg/kg/day. Oral fatty acid supplementation is also often recommended. In some cases systemic retinoids and oral vitamin A can be beneficial. Glucocorticosteroids are usually not effective. SA cannot be cured and lifelong management is required. Affected dogs should not be bred.

Post clipping follicular arrest is characterized by non-inflammatory well-circumscribed area of alopecia confined to a site of previous close clipping or shaving. The reason for the failure of hair regrowth is unclear. "Plush-coated" nordic breeds are predisposed.
Usually the dog will regrew normal hair coat after 7–30 months postclipping. Post-clipping follicular arrest can be an early sign of an endocrinopathy, so if there are presence of other skin and/or systemic abnormalities, then appropriate further diagnostics could be recommended.

English and French Bulldogs

Interdigital furunculosis is characterized by pruritic and/or painful erythematous often fistulated papular/nodular lesions located in the dorsal and/or ventral interdigital webs of dogs. Deep pyogranulomatous inflammation in this case is provoked by reaction to foreign body (contents of ruptured follicles) and secondary bacterial infection. Primary cause in Bulldogs is usually atopic dermatitis and/or wrong anatomical feet conformation. Treatment consist of effective control of primary pathology (in case of AD – mainly systemic cyclosporine and glucocorticosteroids (GCS), topical tacrolimus and GCS; in case of wrong conformation – control of orthopedic pathologies if present, systemic and/or topical GCS to

reduce foreign body reaction, weight loss, protective shoes, decreasing of rubbing in intertrigo areas, and podoplasty if needed) and secondary bacterial infection (long systemical AB courses).

Atopic dermatitis (AD) by definition is genetically-predisposed inflammatory and pruritic skin disease with characteristic clinical features that is associated with IgE antibodies, most commonly directed against environmental allergens. In English and especially French Bulldogs the first symptoms of AD may arise early in life (4-6 months of age) and usually are characterized by recurrent otitis externa and pododermatitis with secondary infections. Pruritus is variable - from almost absent (in some English Bulldogs) to very severe. Treatment is tailored individually and usually consists of a combination of specific (ASIT – allergen-specific immunotherapy) and symptomatic therapy (GCS, cyclosporine, antimicrobials).

Intertrigo is an inflammation of skin folds. Both breeds have multiple folds on the face, tail folds, and sometimes thick skin folds on a dorsal neck. Poor ventilation, increased moisture in the fold (glandular secretions, tears), and rubbing of skin surfaces against each other create environment that favours secondary bacterial and yeast infections. The affected skin is erythematous, pruritic (sometimes painful!), often harbour malodorous exudate and can be eroded. Treatment consist of routine cleaning (wipes are very useful!), topical (rarely systemic) antimicrobial therapy, topical glucocorticoids, and surgical corrections if needed.

Otitis is a common feature in these breeds. More often it is otitis externa due to atopic dermatitis, which is complicated by secondary yeast and bacterial infections, ear canal hyperplasia and otitis media. Recently primary secretory otitis media is reported as a pathology associated with brachycephalic head conformation.

Bull Terrier

Solar (or actinic) dermatosis occurs in white Bull terriers with intense sun exposure. Non-pigmented skin lacks melanin that absorbs UV rays and prevent deeper UV light penetration and actinic damage. Sun exposure may directly damage keratinocytes and cause superficial skin blood vessel dilatation and leakage.
More commonly affected is thinly-haired skin of abdomen and groin. The initial signs of actinic damage are erythema and scaling. With repeated sun exposure, actinic folliculitis/furunculosis, comedones/follicular cyst formation, and dermal fibrosis occur. Chronical sun damage may lead to keratinocyte proliferation, mutagenesis, atypia, and premalignant actinic keratoses, which can progress to invasive squamous cell carcinoma. Sun damaged skin is also more at risk of developing other tumors such as hemangioma, hemangiosarcoma and basal cell carcinoma.
Diagnosis is based on patient's signalment, clinical signs and histopathology results.
Treatment and prevention are achieved by avoidance of excessive sun exposure and applying a waterproof sunscreen with SPF > 25 to exposed areas. Additionally, specially made sunsuits are available for dogs.

Lethal acrodermatitis (LAD) of Bull Terriers is a rare fatal metabolical disorder that is more likely caused by a defect in zinc and copper metabolism. It is characterized clinically by growth retardation, abnormally arched hard palate, progressive hyperkeratotic acrodermatitis, chronic bacterial and yeast skin infections, paronychia, diarrhoea, pneumonia, and abnormal behaviour. Dogs with LAD have been shown to have significantly lower IgA levels than a control group of dogs, which may be the reason for frequent occurrence of microbial infections. This syndrome is inherited as an autosomal recessive trait.
Diagnosis is based on presenting clinical signs and results of histopathology. DNA testing is not available at the moment.
No effective treatment is known. Oral or parenteral treatment with zinc failed to ameliorate the clinical signs of the syndrome. Systemic and/or topical antimicrobial therapy is used to treat secondary infections. Glucocorticoids may help to reduce cutaneous inflammation.

Affected puppies usually die or are euthanized before adulthood (median survival time is 7 months), due to untreatable infections (usually bronchopneumonia and sepsis) and progressive wasting. Both parents of the affected puppies are carriers and should not be bred (as their siblings even if clinically healthy).

Cavalier King Charles Spaniel

Primary secretory otitis media (PSOM) is a disease that has been described almost exclusively in the Cavalier King Charles spaniel (CKCS) and characterized by formation of highly viscous mucus plug in the dog's middle ear and subsequent bulging of the tympanic membrane. Possible cause of PSOM is decreased drainage of the middle ear through the Eustachian (auditory) tube due to certain anatomic differences of CKCS (brachycephalic conformation, greater thickness of the soft palate and reduced cross-sectional area of the nasopharynx).
The majority of dogs will show clinical signs between 3 and 7 years of age. There is no sex predilection. The principal symptoms are moderate to severe pain in the head or neck, guarded and horizontal neck carriage and spontaneous vocalization. Other signs may include neurologic signs (ataxia, facial paralysis, nystagmus, head tilt or seizures), otic pruritus without otitis externa, otitis externa, impaired hearing, and fatigue. None of these clinical signs may be considered pathognomonic for PSOM. Only if a large, bulging pars flaccida is identified the diagnosis is made. However, in many CKCS with PSOM the pars flaccida is flat, and radiographic imaging (eg, CT, MRI) is needed to confirm the diagnosis. Not all CKCS diagnosed with PSOM will have clinical or neurological signs of middle ear disease.
The treatment commonly used consists of performing a myringotomy followed by flushing of the mucus from the bulla. Topical and/or systemic corticosteroids and antibiotics are then administered for otitis media and/or otitis externa. This treatments do not appear to prevent the recurrence of the mucus. Although, mucolytic agents, such as N-acetylcysteine (600 mg every 24 hours) may help to extend the symptom-free time. Tympanostomy tubes or ventral bulla osteotomy may be acceptable alternatives to repeated myringotomy.

Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis (CKSID) is an unique autosomal recessive genetic disease of the Cavalier King Charles spaniel (CKCS) affecting eyes and skin. It is also known as dry eye and curly coat syndrome. Affected puppies have abnormally rough coat at birth, on eyelid opening at 10-14 days symptoms of keratoconjunctivitis sicca become apparent, followed by generalized scaling, hyperkeratosis of footpad margins and distortion and shedding of nails that develops over the next few months. Standing and walking may become difficult and painful. Keratoconjunctivitis sicca is prone to secondary infections and corneal ulceration, that can lead to blindness.
Typical pathological changes are evident in affected puppies from birth, Schirmer tear test, histopathology and/or genetic test are used to confirm a diagnosis of CKSID.
Successful treatment is not possible, although some improvement, particularly of the keratoconjunctivitis sicca, can be obtained with immunomodulatory/lacrimostimulant treatment. Most of affected dogs will be euthanized.
Cavalier King Charles spaniels should be tested for the abnormal gene prior to breeding and two carriers should not be bred together.

Pointer

Acral mutilation syndrome (AMS) is a rare autosomal-recessive genetic sensory neuropathy (progressive degeneration of the sensory neurons in the spinal cord and in peripheral nerves) of dogs that results in absent pain sensation and progressive mutilation of the distal extremities. English Pointers and German shorthaired pointers are predisposed. Affected puppies (more than one in a litter may be affected) are often smaller than unaffected littermates and begin to bite and lick their feet at 3 to 5 months of age and it eventually leads to extensive damage (ulceration, bleeding, and in severe cases auto-amputation of claws, digits and footpads). Single or multiple feet can be affected. Neurological examination usually

reveals acral analgesia - affected animals can walk on their severely mutilated feet without evidence of pain.
Diagnosis is usually based on on distinctive clinical signs in a young dog of a typical breed. Nerve biopsies can also be taken.
Mild affected dogs can be treated with anxiolytic drugs such as diazepam, elizabethan collars, bandages and nursing care of damaged areas, but many cases rapidly deteriorate, requiring euthanasia.
There is no test for carriers of this disorder. Parents and siblings of affected dogs should not be used for breeding.

Exfoliative cutaneous lupus erythematosus (ECLE) is a unique generalized exfoliative dermatitis that is seen exclusively in German shorthaired pointer (GSP) dogs and appears to be quite similar clinically and histologically to exfoliative form of chronic cutaneous lupus erythematosus (CLE) in humans. Pedigree analysis of dogs with ECLE is highly suggestive of an autosomal recessive trait. Genome-wide association study to identify the genomic region harboring the gene involved in the development of ECLE identified a SNP allele on canine chromosome 18 that segregated with the disease. Studies revealed cellular and humoral immune response directed against the epidermal basement membrane.
Common age of onset is 5-7 months and females may be predisposed. Adherent scales, follicular casts, crusting and alopecia first appear on the head (muzzle, pinnae) and back, then generalize and most severely affect pressure points (hocks) and scrotum. Erythema and pruritus are common. Some dogs develop ulceration and depigmentation of the nose. Secondary infections may also be involved. Systemic signs such as pyrexia, letargy and lymphadenopathy are quite common. In advanced cases pain in the joints, lameness, renal disease, and anemia and/or thrombocytopenia may be present.
Definitive diagnosis requires skin histopathology.
Disease usually responds poorly to immunosuppressive therapy (GCS and/or azathioprine, cyclosporine, hydroxychloroquine, adalimumab) and has a guarded prognosis.

Ana Rostaher, DVM, DipECVD

Retrievers

Pyotraumatic dermatitis (acute moist dermatitis, hot spot) is a surface bacterial skin infection (colonisation) caused by trauma. Underlying causes include ectoparasites (fleas, ticks,…), allergies (flea bite hypersensitivity, atopic dermatitis, food allergy), ear infections, anal sac problems, foreign bodies, dirty unkept coat, irritant substances, orthopaedic disorders. Dense-coated dogs are predisposed to developing hot spots, probably because of poor ventilation in their coats.
It is characterized by a circumscribed, well-demarcated, red, moist and erosive/ulcerated area of skin, which is often painful to touch. The development can be very fast and therefore an immediate medical intervention is indicated. It is usually located near or over the primary process (otitis, foreign body, anal sacc infection or impaction), but can be located anywhere in allergic dogs.
The diagnosis of hot spots is quite simple due to the distinctive appearance of this condition. A skin impression smear with subsequent cytological examination should be always performed to characterize the involved infectious agent.
The main differential is the so-called pyotraumatic folliculitis, which is characterized by additional papules as a marker of a deeper process. In this cases glucocorticoids are contraindicated.
Treatment consists of clipping and removing the hair overlying the hot spot and cleaning and disinfecting (chlorhexidin) the surface of the infected skin. Sedation is occasionally needed.
There are many possible ways how to successfully treat a hot spot. The treatment selection depends on the severity of the lesions, underling cause and owners and patients compliance. It consists of topical or systemic glucocorticoids and systemic and/or topical antibacterials (povidone iodine, chlorhexidine, antibiotic creams). The duration is usually between 7-14 days.

The prognosis is excellent, but for uncommon refractory cases the owner should be informed that a further diagnostic work-up (ruling out allergies) would be necessary. There is no specific prophylaxis. Possible ways could be appropriate grooming in dogs with heavy coats, regular parasite control, periodic cleaning of the ears and anal sacs.

Golden Retriever ichthyosis is a quite common keratinization disorders in this breed. The causative PNPLA1 gene was just recently determined by French colleagues. The gene is thought to play a role in lipid organization and metabolism in the upper layers of the epidermis.
Dermatological signs, visible at as early as a few weeks of age, include a mild, moderate or severe generalized scaling, initially with small to large whitish scales and progressively with blackish scales. The scaling can be prominent on the trunk and in some cases ventral hyperpigmentation is present. Some dogs develop secondary bacterial folliculitis, which may lead to pruritus and clinical confusion with allergic skin disease. Although in the authors opinion some dogs can exhibit both diseases. The disease course may wax and wane with periodic bouts of exacerbation and remission.
Histopathology carried out on affected golden retriever dogs shows compact orthokeratotic epidermal hyperkeratosis composed of many layers of completely keratinized epidermal cells and pronounced acanthosis in the epidermis. Additionally, hypergranulosis with increased amounts of keratohyalin and cytoplasmic vacuolic structures in keratinocytes of the (sub)granular layer can be observed.
The gene is thought to play a role in lipid organization and metabolism within the outer epidermis. A genetic test is currently offered by a European company (Antagen; www.antagene.com/en) and can be useful when deciding if an animal should be bread or not. In nonbreeding pets suspected of having the disease, a skin biopsy procedure with examination by an experienced dermatopathologist is sufficient to confirm the clinical diagnosis.
No specific treatment exist, symptomatic treatment depends on the disease severity and may include frequent combing (avoiding trauma and consequent scaling), shampooing and different moisturizers.

Vitiligo is an acquired disease associated with melanocyte destruction, resulting in areas of leukoderma and/or leukotrichia. The exact cause is still not known and it seams that the pathogenesis involves multiple mechanisms. In human medicine it is currently believed that autoimmunity and genetics play a major role in disease development. Additionally, melanocyte damage is thought to arise from oxidative stress (free radicals), missing/deregulated physiologic antioxidant protection, different neural or infectious stimuli. In animals not much is known, but antimelanocyte antibodies have been demonstrated in the serum of dogs.
Typically the lesions involve hypopigmented macules (leukoderma and leukotrichia) and are usually not inflamed. In some cases a transient erythema or scaling are observed (active inflammatory phase of disease?). If the inflammation persists, one should think of additional solar dermatosis. The disease onset is usually in young adults and may progress over months or even years. The term Dudley nose is utilized to describe dogs that lack nasal pigment, generally from birth on. It can be a disqualification for many breeds. Some dogs have a waxing and waning course of nasal hypopigmentation, which is called snow nose or winter nose (usually present during winter). Complete depigmentation is usually not noted in these cases.
Diagnosis is based on signalment, history and clinical examination and may be confirmed by histopathological examination.
In human medicine the first line treatment consist of topical corticosteroids, calcineurin inhibitors, phototherapy and photochemotherapy, whereas vitamin D analogues, targeted phototherapy, oral corticosteroids and surgery should be used as second-line therapies. In veterinary dermatology we do not know much. Possibly effective reported options are tacrolimus ointment and aminoacid L-phenylalanin food supplement (50 mg/kg SID).

Labrador hereditary nasal parakeratosis (HNP) is a keratinization disorder of Labrador retrievers and their crosses. An autosomal-recessive mode of inheritance is suspected. Nasal lesions are first noticed between 6 and 12 months of age and affected dogs are otherwise healthy. Clinical signs include different degress of hyperkeratosis on the dorsal aspect of the nasal planum. It may be associated with mild to

severe loss of pigment and sometimes also the normal ‚comble stone' appearance may be disturbed. In severe cases fissures and ulceration maybe present. The diagnosis is made by clinico-pathological means. The most typical histopathological findings include parakeratotic hyperkeratosis and accumulations of proteinaceous material, the so-called 'serum lakes' between keratinocytes. No curative treatment exists for this life long condition. If necessary, topical moisturizers should be applied (propylene glycol). For severe cases with fissuring, secondary infection should be addressed and the inflammation treated (topical or systemic steroids).

Shar-Pei

Hereditary Cutaneous Mucinosis or hereditary cutaneous hyaluronosis

The Chinese Shar-Pei is known for its distinctive wrinkled, thickened skin, related to a genetic selection desired by breeders. The loose skin and wrinkles covering the head, neck and body become progressively more marked over the first weeks of life, but then tend to decrease, as the dog grows older. It is caused by mucin deposits, which contain an important constituent named hyaluronic acid (HA). In some cases the mucin deposition is excessive and leads to severe skin folding, and/or to the more severe vesicular form characterized by dermal vesicles or bullae, which are filled with a jelly like, clear, viscid substance. On are occasion the skin can tear and ulcerations develop.

Extensive studies by Spanish colleagues proved that these Shar-Pei dogs proved a genetic defect in the metabolism of HA and it is due to an increased activity of the enzyme hyaluran synthetase-2 (HAS2). It was also shown, that these dogs have also elevated HA levels in the blood, which could be potentially used as a disease marker (to the authors knowledge in this moment, no routine lab is offering a test for HA). The main receptor for HA is CD44 and is necessary for its uptake and catabolism. It is expressed in keratinocytes (epidermal and follicular), sebaceous glands, fibroblasts, mast cells and macrophages and maybe of high importance in the pathogenesis of the so called Shar-Pei fever. When fragmented, HA can act as a trigger of the innate immune system and stimulate sterile fever and inflammation, called periodic fever syndrome or Shar-Pei fever.

Mucinosis can be induced by other dermatoses, such as hypothyroidism or in a variety of inflammatory skin diseases, including allergy and pyoderma (Shar-Peis are prone to atopic dermatitis and hypothyroidism).

The diagnosis is usually straightforward by clinico-pathological means.

Some cases do not require treatment, some even spontaneously resolve as the dog ages. A significant deflation occurs following administration of glucocorticoids. Before using them it is important to inform the owner, that the dog can deflate significantly and the dog might loose its typical look. In the author experience NSAIDs can potentially in rare instances also cause deflation. Some authors report pentoxyfilline (10 mg/kg TID) as effective alternative to glucocorticoids, if a long-term therapy is necessary.

Mast cell tumors are common neoplasms of the dog that arise from the connective tissue mast cells. Much work was done in the tumor pathogenesis and recent studies showed that a mutations in the c-kit gene are important for the tumor genesis. They leads to a constant activation and proliferation of mast cells.

Mast cell tumors appear at an avarage age of 8 years, but Shar-Pei dogs have usually an earlier disease onset (average 4 years, 28% less then 2 years) and they are prone to develop multiple lesions. Solitary lesions are can be soft to firm and located dermally or in the subcutis. Often the skin is erythematous. Some can mimic urticarial swellings and some are very diffuse and aggressively growing (often on the limbs).

Diagnosis is straightforward with cytological examination of an impression smear or fine-needle aspiration preparation and histopathological examination. Mast cells are cytologically characterized as discrete, round cells with metachromatically staining intracytoplasmatic granules (which can be also extracellulary due to degranulation). Poorly differentiated mast cells may lack these typical granules, requiring additional diagnostic staining/labeling techniques. Recently, pathologists established a two-tier

histologic grading system (high-grade 7 or more mitotic figures and at least 3 multinucleate cells in 10 high-power fields).
If possible treatment of choice is surgical removal. Some cases with residual microscopic disease need additional radiation therapy, interstitial brachytherapy with iridium-192, chemotherapy or tyrosin kinase inhibitors (toceranib, masitinib).

Chow

Pemphigus foliaceus (PF) is the most common autoimmune disease in the dog. The skin changes are caused by an inflammatory reaction targeting the desmosomes in the epidermis. Differences molecular targets are described in human and canine PF cases. Desmoglein-1 is the main target protein in people, whereas desmocollin-1 was recently shown to be the main target protein in dogs. In most cases a trigger is not identified, but drugs are under discussion as a possible factor. Its development has been also observed in dogs with chronic inflammatory skin disease, such as allergy, but the definitive proof is lacking.
The disease starts usually in middle-aged dogs, but also affected younger or older animals have been reported. The typical lesions are pustules, which can start as papules and in the disease progression crusting is observed. The nasal planum can exhibit variable degrees of depigmentation and the footpads may show crusting and fissuring. Typical predilection sites are head, face and feet and in most of the cases with a striking bilateral symmetry. The onset of clinical signs is usually rapid (within 1-2 weeks), but a protracted course of some months has been reported. The degree of pruritus is variable, ranging from none to severe. Some animals may have fever, are depressed and anorectic. Mucosae are affected very rarely. Some animals may develop secondary pyoderma. The main differentials include dermatophytosis, demodicosis and bacterial folliculitis.
Diagnosis is based on the typical clinical signs and the distribution, presence of acantholytic cells in the cytological examination of impression smears. The diagnosis is confirmed by histopathological examination. If possible an intact pustule should be biopsied. If only crusts are present, it is recommended to include also the crust in the submitted material. Routine hematology and biochemistry are usually unspectacular (neutrophilia), but should be performed to provide a baseline for monitoring if treatment side effects would appear.
The treatment of choice are corticosteroids, usually (methyl-)prednisolone with starting doses of minimally 2 mg/kg SID (up to 6 mg/kg) divided into twice daily or not. The most cases improve within the first 2 weeks, when the drug protocol can be slowly tapered. In severe cases, some authors even prefer the following protocol as first-line treatment, the pulse dosing (10 mg/kg prednisolone SID orally or parenterally for 1-3 doses) can be employed. In refractory cases and cases with severe glucocorticoids side effects, azathioprin (starting dose 1.5-2.5 mg/kg SID) as a glucocorticoid-sparing agent is added. An alternative to azathioprine is chlorambucil (0.1-0.2 mg/kg daily or EOD). Once control is achieved, the goal is to dose these two drugs on the lowest possible dosage on alternate days. Other treatment options include the combination of teytracycline(doxycycline) and niacinamid , cyclosporine and iv immunoglobulins. Topical glucocorticoids and tacrolimus ointment are beneficial for localized lesions.
The prognosis is guarded to good and it depends heavily on the owner. From studies the outcome can be successful from 40% and up to 90% of cases. Some cases even can go into complete remission.

Alopecia X (adrenal hyperplasia-like syndrome, GH responsive alopecia, hyposomatotropism, castration-responsive dermatosis, wooly syndrome, biopsy-responsive alopecia, pseudo-Cushing) represents a hair cycle disorder, in which the normal hair follicle cycling is interrupted at a certain stage. This results in loss of anagen hair follicles and the predominance of telogen follicles and/or increased numbers of catagen. With time these ‘not growing’ hairs are lost, resulting in a slow progressing (over months) non-inflammatory symmetrical alopecia. There are many controversies why this disease develops and the studies on the disease etiopathogenesis are still ongoing, resulting in the formation of two schools. One is pointing toward a problem in the steroid synthesis, the other suggests that the triggering events happen on the hair follicle level including different receptors and local factors.

The diagnosis is made by signalment, clinical examination, ruling out other cause for non-inflammatory alopecias (hypothyroidism, Cushings disease) and is confirmed by histopathological examination of skin biopsies.
The author currently recommends the following management options: benign neglect (as only cosmetic problem), melatonin (3mg for a small dog and 6-12mg for medium and large dogs) and castration (chemical and surgery). A recent study from LA Frank showed partial response to medroxyprogesterone acetate injections (10 mg/kg sc monthly 4 times) without side effects. R Cerundolo and coll. showed that Trilostane may be effective to reverse alopecia. In the authors opinion the risk of potential severe adverse effects is outweighing its current use as a first-line treatment.

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